WEST Search History

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DATE: Wednesday, March 22, 2006

Hide?	Set Name	Query	Hit Count
		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ	
☐	L7	(human or (homo adj sapiens)) same L6	37
☐	L6	(mutant? or variant? or splic$5) same L4	56
☐	L5	(mutant? or variant? or splice) same L4	23
☐	L4	prostaglandin same L3	452
☐	L3	(vector? or express$5 or host)same L2	1050
☐	L2	(gene or sequence or polynucleotide or clone or recombinant) same L1	2025
☐	L1	((cyclooxygenase near1 1) or cyclooxygenase or COX-1 or (Cox near1 1)or (prostaglandin-endoperoxide near1 synthase) or (PG with synthetase)(prostaglandin with G/H with synthase) or (prostaglandin with synthase)or (prostaglandin with synthetase))	12272

END OF SEARCH HISTORY

NiceZyme View of ENZYME: EC 1.14.99.1

Official Name

Prostaglandin-endoperoxide synthase.

Alternative Name(s)

PG synthetase.

Prostaglandin G/H synthase.

Prostaglandin synthase.

Prostaglandin synthetase.

Reaction catalysed

Arachidonate + AH(2) + 2 O(2) <=> prostaglandin H(2) + A + H(2)O

Comment(s)

Acts both as a dioxygenase and as a peroxidase.

Cross-references

Biochemical Pathways; map number(s)	T6 ; U6
BRENDA	1.14.99.1
PUMA2	1.14.99.1
PRIAM enzyme-specific profiles	1.14.99.1
Kyoto University LIGAND chemical database	1.14.99.1
IUBMB Enzyme Nomenclature	1.14.99.1
IntEnz	1.14.99.1
MEDLINE	Find literature relating to 1.14.99.1
MetaCyc	1.14.99.1
UniProtKB/Swiss-Prot	O62664, PGH1_BOVIN; P23219, PGH1_HUMAN; P22437, PGH1_MOUSE; O97554, PGH1_RABIT; Q63921, PGH1_RAT; P05979, PGH1_SHEEP; O62698, PGH2_BOVIN; P70682, PGH2_CAVPO; P27607, PGH2_CHICK; O19183, PGH2_HORSE; P35354, PGH2_HUMAN; Q05769, PGH2_MOUSE; O62725, PGH2_MUSVI; O02768, PGH2_RABIT; P35355, PGH2_RAT; P79208, PGH2_SHEEP;

View entry in original ENZYME format

All UniProtKB/Swiss-Prot entries referenced in this entry, with possibility to download in different formats, align etc.

KEGG ENZYME: 1.14.99.1

Help

Entry	EC 1.14.99.1 Enzyme
Name	prostaglandin-endoperoxide synthase; prostaglandin synthase; prostaglandin G/H synthase; (PG)H synthase; PG synthetase; prostaglandin synthetase; fatty acid cyclooxygenase; prostaglandin endoperoxide synthetase
Class	Oxidoreductases Acting on paired donors with incorporation of molecular oxygen Miscellaneous
Sysname	(5Z,8Z,11Z,14Z)-icosa-5,8,11,14-tetraenoate,hydrogen-donor:oxygen oxidoreductase
Reaction	arachidonate + AH2 + 2 O2 = prostaglandin H2 + A + H2O [RN:R00073 R01590 R01599]
Substrate	arachidonate [CPD:C00219] AH2 [CPD:C00030] O2 [CPD:C00007]
Product	prostaglandin H2 [CPD:C00427] A [CPD:C00028] H2O [CPD:C00001]
Inhibitor	Aspirin [CPD:C01405] Naproxen [CPD:C01517] Sulindac [CPD:C01531] Ibuprofen [CPD:C01588] Piroxicam [CPD:C01608] Diclofenac [CPD:C01690] Diflunisal [CPD:C01691] Ketoprofen [CPD:C01716] Indomethacin [CPD:C01926] Mefenamic acid [CPD:C02168] Tolmetin sodium [CPD:C02328] Fenprofen calcium [CPD:C02539] Meclofenamate sodium [CPD:C02996]
Comment	This enzyme acts both as a dioxygenase and as a peroxidase.
Pathway	PATH: map00590 Arachidonic acid metabolism
Ortholog	KO: K00509 prostaglandin-endoperoxide synthase
Genes	HSA: 5742(PTGS1) 5743(PTGS2) MMU: 19224(Ptgs1) 19225(Ptgs2) RNO: 24693(Ptgs1) 29527(Ptgs2) BTA: 282023(PTGS2) SSC: 397590(PGHS-2) XLA: 446781(ptgs2-prov) DRE: 246226(ptgs1) 246227(ptgs2)
Disease	MIM: 176805 Prostaglandin-endoperoxide synthase 1 (prostaglandin G/H synthase and MIM: 600262 Prostaglandin-endoperoxide synthase 2 (prostaglandin G/H synthase and

=> index bioscience medicine

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 11:31:41 ON 22 MAR 2006

73 FILES IN THE FILE LIST IN STNINDEX

=> s ((cyclooxygenase(w)1) or cyclooxygenase or COX-1 or (Cox(w)1)or (prostaglandin-endoperoxide(w)synthase) or (PG(w)synthetase)or (prostaglandin(w)G/H(w) synthase) or (prostaglandin(w)synthase)or (prostaglandin(w)synthetase))
18642* FILE ADISCTI
232* FILE ADISINSIGHT
75* FILE ADISNEWS
574* FILE AGRICOLA
64* FILE ANABSTR
6* FILE ANTE
1* FILE AQUALINE
95* FILE AQUASCI
244* FILE BIOENG
31551* FILE BIOSIS
217* FILE BIOTECHABS
217* FILE BIOTECHDS
5718* FILE BIOTECHNO
13 FILES SEARCHED...
1891* FILE CABA
27160* FILE CAPLUS
24* FILE CEABA-VTB
86* FILE CIN
489* FILE CONFSCI
3* FILE CROPB
13* FILE CROPU
452* FILE DDFB
11322* FILE DDFU
2895* FILE DGENE
23 FILES SEARCHED...
802* FILE DISSABS
452* FILE DRUGB
0* FILE DRUGMONOG2
12511* FILE DRUGU
302* FILE EMBAL
33401* FILE EMBASE
9964* FILE ESBIOBASE
30 FILES SEARCHED...
671* FILE FEDRIP
0* FILE FOMAD
0* FILE FOREGE
134* FILE FROSTI
82* FILE FSTA
477* FILE GENBANK
43* FILE HEALSAFE
1619* FILE IFIPAT
18* FILE IMSDRUGNEWS
0* FILE IMSPRODUCT
37* FILE IMSRESEARCH
2603* FILE JICST-EPLUS
32* FILE KOSMET
3448* FILE LIFESCI
27441* FILE MEDLINE
107* FILE NIOSHTIC
92* FILE NTIS
7* FILE NUTRACEUT
17* FILE OCEAN
49 FILES SEARCHED...
13429* FILE PASCAL
0* FILE PCTGEN
926* FILE PHAR

```
    37* FILE PHARMAML
     0* FILE PHIC
   119* FILE PHIN
   711* FILE PROMT
  2503* FILE PROUSDDR
     0* FILE PS
     6* FILE RDISCLOSURE
59 FILES SEARCHED...
 23244* FILE SCISEARCH
    51* FILE SYNTHLINE
 22602* FILE TOXCENTER
  7606* FILE USPATFULL
   780* FILE USPAT2
     2* FILE VETB
   213* FILE VETU
     1* FILE WATER
  2437* FILE WPIDS
68 FILES SEARCHED...
    48* FILE WPIFV
  2437* FILE WPINDEX
70 FILES SEARCHED...
   745* FILE IPA
   440* FILE NAPRALERT
   161* FILE NLDB

66 FILES HAVE ONE OR MORE ANSWERS,  73 FILES SEARCHED IN STNINDEX

L1  QUE ((CYCLOOXYGENASE(W) 1) OR CYCLOOXYGENASE OR COX-1 OR (COX(W) 1) OR (PR
    OSTAGLANDIN-ENDOPEROXIDE(W) SYNTHASE) OR (PG(W) SYNTHETASE) OR(PROSTAG
    LANDIN(W) G/H(W) SYNTHASE) OR (PROSTAGLANDIN(W) SYNTHASE) OR (PROSTAGL
    ANDIN(W) SYNTHETASE))

=> d rank
F1     33401* EMBASE
F2     31551* BIOSIS
F3     27441* MEDLINE
F4     27160* CAPLUS
F5     23244* SCISEARCH
F6     22602* TOXCENTER
F7     18642* ADISCTI
F8     13429* PASCAL
F9     12511* DRUGU
F10    11322* DDFU
F11     9964* ESBIOBASE
F12     7606* USPATFULL
F13     5718* BIOTECHNO
F14     3448* LIFESCI
F15     2895* DGENE
F16     2603* JICST-EPLUS
F17     2503* PROUSDDR
F18     2437* WPIDS
F19     2437* WPINDEX
F20     1891* CABA
F21     1619* IFIPAT

=> file f1-f14

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=> s L1

L2 236717 L1

=> s (gene or sequence or polynucleotide or clone or recombinant)(s)L2
L3 12786 (GENE OR SEQUENCE OR POLYNUCLEOTIDE OR CLONE OR RECOMBINANT)(S)
L2

=> dup rem l3

L4 5814 DUP REM L3 (6972 DUPLICATES REMOVED)

=> s (vector# or express? or host)(s)L4
9 FILES SEARCHED...
L5 3424 (VECTOR# OR EXPRESS? OR HOST)(S) L4

=> s prostaglandin# (s)L5
L6 863 PROSTAGLANDIN# (S) L5

=> s (mutant# or variant# or splic?)(s)L6
L7 72 (MUTANT# OR VARIANT# OR SPLIC?)(S) L6

=> s (human or (homo(w)sapiens))(s)L7
11 FILES SEARCHED...
L8 45 (HUMAN OR (HOMO(W) SAPIENS))(S) L7

=> dup rem l8
PROCESSING COMPLETED FOR L8
L9 45 DUP REM L8 (0 DUPLICATES REMOVED)

=> d ibib abs L9 1-45

L9 ANSWER 1 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2006:10667 USPATFULL
TITLE: Lipoxin analogs as novel inhibitors of angiogenesis
INVENTOR(S): Serhan, Charles N., Needham, MA, UNITED STATES
Fierro, Iolanda M., Rio de Janeiro, BRAZIL

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006009521	A1	20060112
APPLICATION INFO.:	US 2005-222458	A1	20050908 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2003-651361, filed on 29 Aug

2003, PENDING Division of Ser. No. US 2002-86609, filed
on 1 Mar 2002, GRANTED, Pat. No. US 6627658

NUMBER DATE
---------- --------
PRIORITY INFORMATION: US 2001-272931P 20010302 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Scott D. Rothenberger, Esq., DORSEY & WHITNEY LLP,
Suite 1500, 50 South Sixth Street, Minneapolis, MN,
55402-1498, US
NUMBER OF CLAIMS: 4
EXEMPLARY CLAIM: 1-8
NUMBER OF DRAWINGS: 7 Drawing Page(s)
LINE COUNT: 2148
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is directed to methods for the prevention or inhibition of angiogenesis. The method is accomplished by the administration of an effective amount of 15-epi-16-(para-fluoro)-phenoxy-lipoxin A.sub.4, LXA.sub.4, 15-epi-LXA.sub.4 or 15-R/S-methyl, LXA.sub.4 and pharmaceutically acceptable salts, esters, amides, carboxylic acids, or prodrugs thereof, to a subject in need thereof. As a consequence of the action of the therapeutic agent, angiogenesis is prevented or inhibited in the subject.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 2 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2006:9955 USPATFULL
TITLE: Identification of tissue/cell specific marker genes and use thereof
INVENTOR(S): Brunner, Andreas, Oberembrach, SWITZERLAND
Hagg, Rupert, Bassesdorf, SWITZERLAND
Tommasini, Roberto, Uster, SWITZERLAND

NUMBER KIND DATE
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PATENT INFORMATION: US 2006008803 A1 20060112
APPLICATION INFO.: US 2003-517756 A1 20030612 (10)
WO 2003-CH379 20030612
20050802 PCT 371 date

NUMBER DATE
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PRIORITY INFORMATION: US 2003-388994P 20020614 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LADAS & PARRY, 26 WEST 61ST STREET, NEW YORK, NY,
10023, US
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1-29
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 4438
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A cartilage array comprises a plurality of different polynucleotide probe spots stably associated with a solid surface of a carrier, whereby each of said spots is made of a unique polynucleotide that corresponds to one specific cartilage marker gene. Said specific cartilage marker genes preferably are at least in part selected from a group of 467 genes that could be shown to be cartilage related.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 3 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:306409 USPATFULL
TITLE: Epicatechin for hypertension treatment
INVENTOR(S): Romanczyk, Leo J. JR., Hackettstown, NJ, UNITED STATES
Schmitz, Harold H., Bethesda, MD, UNITED STATES
PATENT ASSIGNEE(S): Mars, Incorporated, McLean, VA, UNITED STATES (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2005267052 A1 20051201
APPLICATION INFO.: US 2005-166499 A1 20050624 (11)
RELATED APPLN. INFO.: Continuation of Ser. No. US 2004-795552, filed on 8 Mar 2004, PENDING Continuation of Ser. No. US 2004-770969, filed on 2 Feb 2004, GRANTED, Pat. No. US 6900241 Division of Ser. No. US 2002-127817, filed on 22 Apr 2002, PENDING Continuation of Ser. No. US 2000-717893, filed on 21 Nov 2000, GRANTED, Pat. No. US 6670390 Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NADA JAIN, P.C., 560 White Plains Road, Suite 460, Tarrytown, NY, 10591, US
NUMBER OF CLAIMS: 30
EXEMPLARY CLAIM: 1-208
NUMBER OF DRAWINGS: 242 Drawing Page(s)
LINE COUNT: 4531
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are flavanols, e.g. epicatechin, compositions such as pharmaceutical compositions containing an effective amount of flavanols, e.g. epicatechin, and methods of treatment or prevention of hypertension using the same.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 4 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:298899 USPATFULL
TITLE: Non-cytotoxic oriP replicon
INVENTOR(S): Sugden, Bill, Madison, WI, UNITED STATES
Wang, Jindong, Madison, WI, UNITED STATES
Kennedy, Gregory Dean, Madison, WI, UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2005260564 A1 20051124
APPLICATION INFO.: US 2004-848976 A1 20040519 (10)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SCHWEGMAN, LUNDBERG, WOESSNER & KLUTH, P.A., P.O. BOX 2938, MINNEAPOLIS, MN, 55402-0938, US
NUMBER OF CLAIMS: 51
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 2893
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a vector encoding a derivative of EBNA-1 that is not cytotoxic when expressed efficiently in cells, which supports extrachromosomal replication, maintenance and transcription from extrachromosomal oriP containing vectors but does not substantially activate transcription from host cell genes. Also provided is a vector having oriP and encoding a derivative of EBNA-1. The vectors of the invention may be employed in vitro and in gene therapy.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 5 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:268112 USPATFULL
TITLE: Human COX-1 alternatively spliced variants and methods of using same
INVENTOR(S): Liang, Yanbin, Irvine, CA, UNITED STATES
Woodward, David F., Lake Forest, CA, UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2005233429 A1 20051020
APPLICATION INFO.: US 2003-663377 A1 20030915 (10)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: McDERMOTT, WILL & EMERY, 7th Floor, 4370 La Jolla Village Drive, San Diego, CA, 92122, US
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Page(s)
LINE COUNT: 3751
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides an isolated polypeptide containing the amino acid sequence of SEQ ID NO: 14, 16, 18, 20, 22 or 24. The invention also provides an isolated polypeptide containing a) an amino acid sequence having at least 50% amino acid identity with SEQ ID NO: 10, and b) an amino acid sequence selected from SEQ ID NOS: 14, 16, 18, 20, 22, or 24; or a conservative variant thereof. The invention further provides an isolated polypeptide containing the amino acid sequence of SEQ ID NO: 2, 4, 6, or 8. The invention also provides a method for identifying a compound that modulates a COX-1 variant by contacting an isolated COX-1 variant or a COX-1 variant over-expressed in a genetically engineered cell with a compound and determining the level of an indicator which correlates with modulation of a COX-1 variant, where an alteration in the level of the indicator as compared to a control level indicates that the compound is a compound that modulates the COX-1 variant.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 6 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:241167 USPATFULL
TITLE: Treatment of periodontal disease
INVENTOR(S): Romanczyk, Leo J. JR., Hackettstown, NJ, UNITED STATES
Schmitz, Harold H., Bethesda, MD, UNITED STATES
PATENT ASSIGNEE(S): Mars, Incorporated, McLean, VA, UNITED STATES (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005209171	A1	20050922
APPLICATION INFO.:	US 2004-4677	A1	20041203 (11)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2004-795552, filed on 8 Mar 2004, PENDING Continuation of Ser. No. US 2004-770969, filed on 2 Feb 2004, GRANTED, Pat. No. US 6900241 Division of Ser. No. US 2002-127817, filed on 22 Apr 2002, PENDING Continuation of Ser. No. US 2001-776649, filed on 5 Feb 2001, GRANTED, Pat. No. US 6638971 Continuation of Ser. No. US 1997-831245, filed on 2 Apr 1997, GRANTED, Pat. No. US 6297273 Continuation-in-part of Ser. No. US 1996-631661, filed on 2 Apr 1996, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NADA JAIN, P.C., 560 White Plains Road, Suite 460, Tarrytown, NY, 10591, US
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1-208
NUMBER OF DRAWINGS: 242 Drawing Page(s)
LINE COUNT: 4540
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed and claimed are cocoa extracts, compounds, combinations thereof and compositions containing the same, such as polyphenols or procyanidins, methods for preparing such extracts, compounds and compositions, as well as uses for them, especially a polymeric compound of the formula A.sub.n, wherein A is a monomer of the formula: ##STR1## wherein n is an integer from 2 to 18, such that there is at least one terminal monomeric unit A, and one or a plurality of additional monomeric units; R is 3-(.alpha.)-OH, 3-(.beta.)-OH, 3-(.alpha.)-O-sugar, or 3-(.beta.)-O-sugar; bonding between adjacent monomers takes place at positions 4, 6 or 8; a bond of an additional monomeric unit in position 4 has alpha or beta stereochemistry; X, Y and

Z are selected from the group consisting of monomeric unit A, hydrogen, and a sugar, with the provisos that as to the at least one terminal monomeric unit, bonding of the additional monomeric unit thereto (the bonding of the additional monomeric unit adjacent to the terminal monomeric unit) is at position 4 and optionally Y=Z=hydrogen; the sugar is optionally substituted with a phenolic moiety, at any position on the sugar, for instance via an ester bond, and pharmaceutically acceptable salts or derivatives thereof (including oxidation products).

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 7 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:189432 USPATFULL
TITLE: Nucleic acid molecule encoding homer 1B protein
INVENTOR(S): Worley, Paul F., Baltimore, MD, UNITED STATES
Tu, Jian Cheng, Baltimore, MD, UNITED STATES
Xiao, Bo, Ellicott City, MD, UNITED STATES
Leahy, Daniel, Baltimore, MD, UNITED STATES
Beneken, Jutta, Baltimore, MD, UNITED STATES
Lanahan, Anthony A., Baltimore, MD, UNITED STATES
Brakeman, Paul R., Baltimore, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005164344	A1	20050728
APPLICATION INFO.:	US 2004-8889	A1	20041210 (11)

RELATED APPLN. INFO.: Division of Ser. No. US 2002-192381, filed on 9 Jul 2002, GRANTED, Pat. No. US 6864083 Division of Ser. No. US 1999-377285, filed on 18 Aug 1999, GRANTED, Pat. No. US 6720175

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-138494P	19990610 (60)
	US 1999-138493P	19990610 (60)
	US 1999-138426P	19990610 (60)
	US 1998-97334P	19980818 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Lisa A. Haile, J.D., Ph.D., GRAY CARY WARE & FREIDENRICH LLP, 4365 Executive Drive, Suite 1100, San Diego, CA, 92121-2133, US
NUMBER OF CLAIMS: 6
EXEMPLARY CLAIM: 1-7
NUMBER OF DRAWINGS: 55 Drawing Page(s)
LINE COUNT: 7396
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method is provided for identifying a compound that modulates a cellualr response associated with Homer and mediated by a cell-surface or an intracellular receptor. A method is further provided for identifying a compound that modulates receptor activated calcium mobilization associated with Homer. A method is provided for identifying a compound that inhibits Homer protein activity based on the crystal structure coordinates of Homer protein binding domain. A method is also provided for identifying a compound that affects the formation of cell surface receptors into clusters. Also provided are nucleic acids encoding Homer proteins as well as Homer proteins, and Homer interacting proteins.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 8 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:189427 USPATFULL
TITLE: Compositions and methods for dehydration and cyclization of peptides, synthetic compounds, and lantibiotics
INVENTOR(S): van der Donk, Willem A., Champaign, IL, UNITED STATES
Xie, Lili, Brookline, MA, UNITED STATES
Chatterjee, Champak, Urbana, IL, UNITED STATES
Paul, Moushumi, Urbana, IL, UNITED STATES

NUMBER KIND DATE

PATENT INFORMATION: US 2005164339 A1 20050728
APPLICATION INFO.: US 2005-34275 A1 20050112 (11)

NUMBER DATE

PRIORITY INFORMATION: US 2004-536140P 20040112 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: GREENLEE WINNER AND SULLIVAN P C, 4875 PEARL EAST CIRCLE, SUITE 200, BOULDER, CO, 80301, US
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 80 Drawing Page(s)
LINE COUNT: 6180
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Lantibiotics are synthesized on ribosomes as prepeptides and post-translationally modified to a mature form. These modifications include dehydrations and cyclizations. Compounds and related methods of generating compounds, modified by dehydration, cyclization, or dehydration and cyclization, are disclosed. The disclosure includes in vitro approaches to effecting dehydration and cyclization leading to production of biologically active compounds such as lantibiotics and variants thereof. Synthetic variants and methods including combinatorial approaches for generating diverse lantibiotics and other compounds are disclosed. The invention has broad potential for applications including food, agricultural, and medical industries.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 9 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:111513 USPATFULL
TITLE: Identification of ovarian cancer tumor markers and therapeutic targets
INVENTOR(S): Jazaeri, Amir A, Charlottsville, VA, UNITED STATES
Boyd, Jeffrey, Dobbs Ferry, NY, UNITED STATES
Liu, Edison T, Cuscaden Walk, SINGAPORE

NUMBER KIND DATE

PATENT INFORMATION: US 2005095592 A1 20050505
APPLICATION INFO.: US 2003-505680 A1 20030213 (10)
WO 2003-US4688 20030213

NUMBER DATE

PRIORITY INFORMATION: US 2003-357031P 20020213 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KLARQUIST SPARKMAN, LLP, 121 S.W. SALMON STREET, SUITE #1600, ONE WORLD TRADE CENTER, PORTLAND, OR, 97204-2988, US
NUMBER OF CLAIMS: 57
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 20 Drawing Page(s)
LINE COUNT: 6049
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present disclosure provides methods for classifying ovarian tumors into BRCA1-type, BRCA2-type or non-BRCA-type tumor types by measuring expression levels of a plurality of disclosed ovarian tumor markers. The markers disclosed herein are useful in the diagnosis, staging, detection, and/or treatment of ovarian cancer. Also provided are methods of selecting a treatment regimen by selecting the tumor type. Ovarian cancer-linked logarithmic expression ratios and kits for diagnosis, staging, and detection of ovarian cancer using are also provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 10 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:62962 USPATFULL
TITLE: Methods for identifying and using maintenance genes
INVENTOR(S): Warrington, Janet A., Los Altos, CA, UNITED STATES
Mahadevappa, Mamatha, Fremont, CA, UNITED STATES
Nair, Archana, Santa Clara, CA, UNITED STATES
PATENT ASSIGNEE(S): Affymetrix, INC., Santa Clara, CA (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005053998	A1	20050310
APPLICATION INFO.:	US 2004-968652	A1	20041018 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 2000-693204, filed on 19 Oct 2000, GRANTED, Pat. No. US 6841348

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-161000P	19991021 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: AFFYMETRIX, INC, ATTN: CHIEF IP COUNSEL, LEGAL DEPT., 3380 CENTRAL EXPRESSWAY, SANTA CLARA, CA, 95051
NUMBER OF CLAIMS: 2
EXEMPLARY CLAIM: 1
LINE COUNT: 1142
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides methods for discovering maintenance genes and for using maintenance genes. In one embodiment, the expression of at least three maintenance genes are measured and used as reference (or control) for comparing the expression of target genes in two or more biological samples.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 11 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2005:6834 USPATFULL
TITLE: Methods for identifying and using maintenance genes
INVENTOR(S): Warrington, Janet A., Los Altos, CA, United States
Mahadevappa, Mamatha, Cupertino, CA, United States
Nair, Archana, The Woodlands, TX, United States
PATENT ASSIGNEE(S): Affymetrix, Inc., Santa Clara, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6841348	B1	20050111
APPLICATION INFO.:	US 2000-693204		20001019 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-161000P	19991021 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Johannsen, Diana B.
LEGAL REPRESENTATIVE: Wells, Sandra E., McGarrigle, Philip L.
NUMBER OF CLAIMS: 2
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 0 Drawing Figure(s); 0 Drawing Page(s)
LINE COUNT: 853
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides methods for discovering maintenance genes and for using maintenance genes. In one embodiment, the expression of at least three maintenance genes are measured and used as reference (or control) for comparing the expression of target genes in two or more biological samples.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 12 OF 45 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 2005-0138733 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Gastrin promotes human colon cancer cell growth via CCK-2 receptor-mediated cyclooxygenase-2 induction and prostaglandin E.sub.2 production
AUTHOR: COLUCCI Rocchina; BLANDIZZI Corrado; TANINI Marzia; VASSALLE Cristina; BRESCHI Maria Cristina; DEL TACCA Mario
CORPORATE SOURCE: Interdepartmental Center for Research in Clinical Pharmacology and Experimental Therapeutics, University of Pisa, Pisa, Italy; Institute of Clinical Physiology, National Research Council, Pisa, Italy
SOURCE: British journal of pharmacology, (2005), 144(3), 338-348, 37 refs.
ISSN: 0007-1188 CODEN: BJPCBM
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United Kingdom
LANGUAGE: English
AVAILABILITY: INIST-4509, 354000126282290060
AN 2005-0138733 PASCAL
CP Copyright .COPYRGT. 2005 INIST-CNRS. All rights reserved.
AB 1 The present study investigates the effects of gastrin-17 on ***human*** colon cancer HT-29 cells to examine whether gastrin receptor (CCK-2), ***cyclooxygenase*** (***COX*** - ***1*** , COX-2) isoforms and ***prostaglandin*** receptor pathways interact to control cell growth. 2 Reverse transcription (RT)-polymerase chain reaction (PCR) analysis demonstrated that HT-29 cells are endowed with the naive ***expression*** of CCK-2 receptor (short ***splice*** ***variant***), ***COX*** - ***1*** , COX-2 and ***prostaglandin*** EP.sub.4 receptor, but not gastrin. 3 Gastrin-17 significantly promoted cell growth and DNA synthesis. Both these stimulating effects were abolished by L-365,260 or GV150013 (CCK-2 receptor antagonists), but were unaffected by SC-560 (***COX*** - ***1*** inhibitor). L-745,337 (COX-2 inhibitor) or AH-23848B (EP.sub.4 receptor antagonist) partly reversed gastrin-17-induced cell growth, while they fully antagonized the enhancing action on DNA synthesis. 4 HT-29 cells responded to gastrin-17 with a significant increase in ***prostaglandin*** E.sub.2 release. This enhancing effect was completely counteracted by L-365,260, GV150013 or L-745,337, while it was insensitive to cell incubation with SC-560. 5 Exposure of HT-29 cells to gastrin-17 was followed by an increased phosphorylation of both extracellular regulated kinases (ERK-1/ERK-2) and Akt. Moreover, gastrin-17 enhanced the transcriptional activity of COX-2 ***gene*** promoter and stimulated COX-2 ***expression*** . These latter effects were antagonized by L-365,260 or GV150013, and could be blocked also by PD98059 (inhibitor of ERK-1/ERK-2 phosphorylation) or wortmannin (inhibitor of phosphatidylinositol 3-kinase). Analogously, gastrin-17-induced ***prostaglandin*** E.sub.2 release was prevented by PD98059 or wortmannin. 6 The present results suggest that (a) in ***human*** colon cancer cells endowed with CCK-2 receptors, gastrin-17 is able to enhance the transcriptional activity of COX-2 ***gene*** through the activation of ERK-1/ERK-2- and phosphatidylinositol 3-kinase/Akt-dependent pathways; (b) these stimulant actions lead to downstream increments of COX-2 ***expression*** , followed by ***prostaglandin*** E.sub.2 production and EP.sub.4 receptor activation; (c) the recruitment of COX-2/ ***prostaglandin*** pathways contributes to the growth-promoting actions exerted by gastrin-17.

L9 ANSWER 13 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:480771 CAPLUS
DOCUMENT NUMBER: 144:1111
TITLE: Expression of cyclooxygenase-2 mRNA and identification of its splice variant in human myometrium obtained from women in labor
AUTHOR(S): Huang, Yinping; Ye, Duyun; Wu, Ping; Huang, Yanjun; Zhang, Li; Zhou, Xiaoyan; Huang, Yunfeng; Yuan, Ping; Zhang, Daijuan; Wan, Jingyuan
CORPORATE SOURCE: Department of Obstetrics and Gynecology, First

Affiliated Hospital, Wenzhou Medical College, Wenzhou, 325000, Peop. Rep. China
SOURCE: Journal of Huazhong University of Science and Technology, Medical Sciences (2005), 25(1), 5-7
CODEN: JHUSAW; ISSN: 1672-0733
PUBLISHER: Huazhong University of Science and Technology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In order to investigate the expression of cyclooxygenase-2 (COX-2) in human lower segments of myometrium obtained from women in labor and those not in labor and identify the splicing variant of COX-2, reverse transcriptase-polymerase chain reaction (RT-PCR) was used to detect the expression of COX-2. The primers were designed and synthesized according to the sequence of rat COX-2 splice variant which was discovered firstly by us. Then the splicing variant of COX-2 in human myometrium from woman in labor was identified, cloned into vector and sequenced. The results showed that the expression of COX-2 mRNA was lower in human myometrium obtained from women who were not in labor than that in labor women and a new band of COX-2 was obtained in myometrium from labor woman. The fragment included an unspliced intron, which pitched between exons 7 and 8. It was suggested that COX-2 gene was not only expressed highly in human myometrium from woman in labor, but also produced splicing variant by alternative splicing.
REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L9 ANSWER 14 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:292180 USPATFULL
TITLE: Diagnostics and therapeutics for restenosis
INVENTOR(S): Crossman, David C., Sheffield, UNITED KINGDOM
Duff, Gordon W., South Yorkshire, UNITED KINGDOM
Francis, Sheila E., Sheffield, UNITED KINGDOM
Kornman, Kenneth S., San Antonio, TX, UNITED STATES
Stephenson, Katherine, San Antonio, TX, UNITED STATES
PATENT ASSIGNEE(S): Interleukin Genetics, Inc. (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004229264	A1	20041118
APPLICATION INFO.:	US 2004-823197	A1	20040412 (10)

RELATED APPLN. INFO.: Division of Ser. No. US 2000-578534, filed on 24 May 2000, GRANTED, Pat. No. US 6720141 Continuation-in-part of Ser. No. US 1999-431352, filed on 1 Nov 1999, GRANTED, Pat. No. US 6524795 Continuation-in-part of Ser. No. US 1999-320395, filed on 26 May 1999, ABANDONED Continuation-in-part of Ser. No. US 1997-813456, filed on 10 Mar 1997, GRANTED, Pat. No. US 6210877
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MINTZ, LEVIN, COHN, FERRIS, GLOVSKY, AND POPEO, P.C., ONE FINANCIAL CENTER, BOSTON, MA, 02111
NUMBER OF CLAIMS: 10
EXEMPLARY CLAIM: CLM-001-7
NUMBER OF DRAWINGS: 19 Drawing Page(s)
LINE COUNT: 5080
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and kits for determining whether a subject has or is predisposed to developing restenosis are provided.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 15 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:239679 USPATFULL
TITLE: Gene markers useful for detecting skin damage in response to ultraviolet radiation
INVENTOR(S): Blumenberg, Miroslav, New York, NY, UNITED STATES
PATENT ASSIGNEE(S): New York University, New York, NY, UNITED STATES (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 2004185485 A1 20040923
APPLICATION INFO.: US 2004-775875 A1 20040210 (10)
RELATED APPLN. INFO.: Division of Ser. No. US 2001-947870, filed on 6 Sep 2001, PENDING

NUMBER DATE

PRIORITY INFORMATION: US 2000-231454P 20000908 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WILMER CUTLER PICKERING HALE AND DORR LLP, 60 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 97
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 10307
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The cellular response to ultraviolet radiation exposure has been characterized on the molecular level through the use of high density gene array technology. Nucleic acid molecules and protein molecules, the expression of which are repressed or induced in response to ultraviolet radiation exposure, are identified according to a temporal pattern of altered expression post ultraviolet radiation exposure. Methods are disclosed that utilized these ultraviolet radiation-regulated molecules as markers for ultraviolet radiation exposure. Other screening methods of the invention are designed for the identification of compounds that modulate the response of a cell to ultraviolet radiation exposure. The invention also provides compositions useful for drug screening or pharmaceutical purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 16 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:184452 USPATFULL
TITLE: Method for determining skin stress or skin ageing in vitro
INVENTOR(S): Petersohn, Dirk, Koeln, GERMANY, FEDERAL REPUBLIC OF
Conradt, Marcus, Pretoria, SOUTH AFRICA
Hofmann, Kay, Koeln, GERMANY, FEDERAL REPUBLIC OF

NUMBER KIND DATE

PATENT INFORMATION: US 2004142335 A1 20040722
APPLICATION INFO.: US 2003-450797 A1 20030917 (10)
WO 2001-EP15178 20011220

NUMBER DATE

PRIORITY INFORMATION: DE 2001-100121 20010103
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WOODCOCK WASHBURN LLP, ONE LIBERTY PLACE, 46TH FLOOR, 1650 MARKET STREET, PHILADELPHIA, PA, 19103
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 11268
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention relates to a method for determining skin stress and/or skin ageing in humans or animals in vitro, test kits and biochips for determining skin stress and/or skin ageing, and the use of proteins, mRNA molecules or fragments of proteins or mRNA molecules as skin stress and/or ageing markers. The invention also relates to a test method for demonstrating the effectiveness of cosmetic or pharmaceutical active ingredients against skin stress and/or skin ageing, a screening method for identifying cosmetic or pharmaceutical active ingredients against skin stress and/or skin ageing, and a method for producing a cosmetic and/or pharmaceutical preparation against skin stress and/or skin ageing. The invention further relates to a cosmetic or pharmaceutical

preparation against skin stress and/or skin ageing.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 17 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:101093 USPATFULL
TITLE: Methods of diagnosis of bladder cancer, compositions and methods of screening for modulators of bladder cancer
INVENTOR(S): Mack, David H., Menlo Park, CA, UNITED STATES
Aziz, Natasha, Palo Alto, CA, UNITED STATES
PATENT ASSIGNEE(S): Eos Biotechnology, Inc., South San Francisco, CA, UNITED STATES, 94080-7019 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004076955	A1	20040422
APPLICATION INFO.:	US 2002-188832	A1	20020702 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-372246P	20020412 (60)
	US 2001-350666P	20011113 (60)
	US 2001-343705P	20011108 (60)
	US 2001-310099P	20010803 (60)
	US 2001-302814P	20010703 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOWREY SIMON ARNOLD & WHITE, LLP, BOX 34, 301 RAVENSWOOD AVE., MENLO PARK, CA, 94025
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 27357
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described herein are genes whose expression are up-regulated or down-regulated in bladder cancer. Also described are such genes whose expression is further up-regulated or down-regulated in drug-resistant bladder cancer cells. Related methods and compositions that can be used for diagnosis, prognosis, or treatment of bladder cancer are disclosed. Also described herein are methods that can be used to identify modulators of bladder cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 18 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:50862 USPATFULL
TITLE: Wound healing biomarkers
INVENTOR(S): Burslem, Martyn Frank, Sandwich, UNITED KINGDOM
Johnson, Claire Michelle, Sandwich, UNITED KINGDOM
Cooper, Lisa, London, UNITED KINGDOM
Martin, Paul, London, UNITED KINGDOM

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004038292	A1	20040226
APPLICATION INFO.:	US 2002-175184	A1	20020618 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	GB 2001-14869	20010618
	US 2001-305346P	20010713 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: PFIZER INC., PATENT DEPARTMENT, MS8260-1611, EASTERN POINT ROAD, GROTON, CT, 06340
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 26 Drawing Page(s)
LINE COUNT: 67123
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention provides biomarkers such as genes and the corresponding mRNA transcripts or protein products that are identified as being involved in wound healing processes. Also provided are methods for identification of compounds useful for the treatment of wounds, wound healing disorders or inflammation and compounds identified by such methods. Methods are provided for monitoring the progress of wound healing and for identification of individuals with wound healing disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 19 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:25211 USPATFULL
TITLE: Combination of an allosteric carboxylic inhibitor of matrix metalloproteinase-13 with celecoxib or valdecoxib
INVENTOR(S): Roark, William Howard, Ann Arbor, MI, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004019053	A1	20040129
APPLICATION INFO.:	US 2003-619662	A1	20030715 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-396903P	20020717 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: WARNER-LAMBERT COMPANY, 2800 PLYMOUTH RD, ANN ARBOR, MI, 48105
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 1
LINE COUNT: 8040
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention provides a combination, comprising an allosteric carboxylic inhibitor of MMP-13, or a pharmaceutically acceptable salt thereof, with celecoxib, or a pharmaceutically acceptable salt thereof, or valdecoxib, or a pharmaceutically acceptable salt thereof. This invention also provides a method of treating a disease that is responsive to inhibition of MMP-13 and cyclooxygenase-2, comprising administering to a patient suffering from such a disease the invention combination comprising an allosteric carboxylic inhibitor of MMP-13, or a pharmaceutically acceptable salt thereof, with celecoxib, or a pharmaceutically acceptable salt thereof, or valdecoxib, or a pharmaceutically acceptable salt thereof. This invention also provides a pharmaceutical composition, comprising the invention combination comprising an allosteric carboxylic inhibitor of MMP-13, or a pharmaceutically acceptable salt thereof, with celecoxib, or a pharmaceutically acceptable salt thereof, or valdecoxib, or a pharmaceutically acceptable salt thereof, and a pharmaceutically acceptable carrier, diluent, or excipient.

The invention combination may also be further combined with other pharmaceutical agents depending on the disease being treated.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 20 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:18738 USPATFULL
TITLE: Cardiotoxin molecular toxicology modeling
INVENTOR(S): Mendrick, Donna, Gaithersburg, MD, UNITED STATES
Porter, Mark, Gaithersburg, MD, UNITED STATES
Johnson, Kory, Gaithersburg, MD, UNITED STATES
Higgs, Brandon, Gaithersburg, MD, UNITED STATES
Castle, Arthur, Gaithersburg, MD, UNITED STATES
Elashoff, Michael, Gaithersburg, MD, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004014040	A1	20040122

APPLICATION INFO.: US 2002-191803 A1 20020710 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-303819P	20010710 (60)
	US 2001-305623P	20010717 (60)
	US 2002-369351P	20020403 (60)
	US 2002-377611P	20020506 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: MORGAN LEWIS & BOCKIUS LLP, 1111 PENNSYLVANIA AVENUE NW, WASHINGTON, DC, 20004
NUMBER OF CLAIMS: 59
EXEMPLARY CLAIM: 1
LINE COUNT: 15812
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention is based on the elucidation of the global changes in gene expression and the identification of toxicity markers in tissues or cells exposed to a known cardiotoxin. The genes may be used as toxicity markers in drug screening and toxicity assays. The invention includes a database of genes characterized by toxin-induced differential expression that is designed for use with microarrays and other solid-phase probes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 21 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:7410 USPATFULL
TITLE: Method and composition for detection and treatment of breast cancer
INVENTOR(S): Su, Yan A., Bethesda, MD, UNITED STATES
Yang, Jun, Hinsdale, IL, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004005644	A1	20040108
APPLICATION INFO.:	US 2003-373801	A1	20030227 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-359999P	20020228 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: DORSEY & WHITNEY LLP, 1001 PENNSYLVANIA AVENUE, N.W., SUITE 400 SOUTH, WASHINGTON, DC, 20004
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 6110
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method for the detection of breast cancer using breast by measuring expression levels of breast cancer specific marker (BCSM) genes, and in particular the level of polynucleotides transcribed from and polypeptides encoded by the BCSM genes. The present invention also provide a method for the treatment and/or prevention of breast cancer by modulating the activity of BCSM genes or the products of BCSM genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 22 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:7329 USPATFULL
TITLE: Methods of diagnosis of ovarian cancer, compositions and methods of screening for modulators of ovarian cancer
INVENTOR(S): Mack, David H., Menlo Park, CA, UNITED STATES
Gish, Kurt C., San Francisco, CA, UNITED STATES
PATENT ASSIGNEE(S): Eos Biotechnology, Inc., South San Francisco, CA (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 2004005563 A1 20040108
APPLICATION INFO.: US 2002-173999 A1 20020617 (10)

NUMBER DATE
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PRIORITY INFORMATION: US 2002-372246P 20020412 (60)
US 2001-350666P 20011113 (60)
US 2001-315287P 20010827 (60)
US 2001-299234P 20010618 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834
NUMBER OF CLAIMS: 24
EXEMPLARY CLAIM: 1
LINE COUNT: 32540
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Described herein are genes whose expression are up-regulated or down-regulated in ovarian cancer. Related methods and compositions that can be used for diagnosis and treatment of ovarian cancer are disclosed. Also described herein are methods that can be used to identify modulators of ovarian cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 23 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:263898 USPATFULL
TITLE: Methods for compositions for in vivo gene delivery
INVENTOR(S): Debs, Robert James, Mill Valley, CA, United States
Zhu, Ning, El Cerrito, CA, United States
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

NUMBER KIND DATE
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PATENT INFORMATION: US 6806084 B1 20041019
APPLICATION INFO.: US 1998-90030 19980610 (9)
RELATED APPLN. INFO.: Continuation of Ser. No. US 1994-246376, filed on 19 May 1994, now patented, Pat. No. US 5827703
Continuation of Ser. No. US 1992-992687, filed on 17 Dec 1992, now abandoned Continuation-in-part of Ser. No. US 1992-927200, filed on 6 Aug 1992, now abandoned Continuation-in-part of Ser. No. US 1992-894498, filed on 4 Jun 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Nguyen, Dave T.
LEGAL REPRESENTATIVE: Townsend & Townsend & Crew LLP
NUMBER OF CLAIMS: 32
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 60 Drawing Figure(s); 71 Drawing Page(s)
LINE COUNT: 2466
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel methods and compositions are provided for introducing a gene capable of modulating the genotype and phenotype into two or more tissues following systemic administration. The gene can be introduced into a mammalian host by way of an expression vector either as naked DNA or complexed to lipid carriers, particularly cationic lipid carriers. Multiple individual tissues can be transfected using naked DNA. Using a DNA:lipid carrier complex, multiple tissues and cell types can be transfected. The techniques and compositions find use in the palliation or treatment of any of a variety of genetic-based disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 24 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:90618 USPATFULL
TITLE: Diagnostics and therapeutics for restenosis

INVENTOR(S): Crossman, David C., Sheffield, UNITED KINGDOM
Duff, Gordon W., South Yorkshire, UNITED KINGDOM
Francis, Sheila E., Sheffield, UNITED KINGDOM
Kornman, Kenneth S., San Antonio, TX, United States
Stephenson, Katherine, San Antonio, TX, United States
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6720141	B1	20040413
APPLICATION INFO.:	US 2000-578534		20000524 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-431352, filed on 1 Nov 1999
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Myers, Carla J.
LEGAL REPRESENTATIVE: Elrifi, Esq., Ivor R., Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.
NUMBER OF CLAIMS: 14
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 10 Drawing Figure(s); 19 Drawing Page(s)
LINE COUNT: 4958
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and kits for determining whether a subject has or is predisposed to developing restenosis are provide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 25 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2004:66006 USPATFULL
TITLE: DNA array sequence selection
INVENTOR(S): Lorenz, Matthias, Bethesda, MD, United States
PATENT ASSIGNEE(S): The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6706867	B1	20040316
APPLICATION INFO.:	US 2000-741238		20001219 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Horlick, Kenneth R.
ASSISTANT EXAMINER: Wilder, Cynthia
LEGAL REPRESENTATIVE: Leydig, Voit & Mayer, Ltd.
NUMBER OF CLAIMS: 8
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 29 Drawing Page(s)
LINE COUNT: 23532
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides methods and compositions for the construction of custom cDNA microarrays. In particular, the methods involve the selection of relevant clusters based on knowledge and expression patterns using public database information and the identification of the best representative cDNA clones within the selected cluster. The methods facilitate the construction of custom microarrays suitable for use in any biotechnological art. In preferred embodiments, the present invention provides the the ImmunoChip.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 26 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:250978 USPATFULL
TITLE: Diagnostics and therapeutics for cardiovascular disease
INVENTOR(S): Francis, Sheila E., Sheffield, UNITED KINGDOM
Crossman, David C., Sheffield, UNITED KINGDOM
Duff, Gordon W., Sheffield, UNITED KINGDOM
Kornman, Kenneth S., Newton, MA, UNITED STATES
Stephenson, Katherine, San Antonio, TX, UNITED STATES

PATENT ASSIGNEE(S): Interleukin Genetics, Inc., Waltham, MA (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003175764	A1	20030918
APPLICATION INFO.:	US 2002-320360	A1	20021213 (10)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1999-431352, filed on 1 Nov 1999, GRANTED, Pat. No. US 6524795 Continuation-in-part of Ser. No. US 1999-320395, filed on 26 May 1999, ABANDONED Continuation-in-part of Ser. No. US 1997-813456, filed on 10 Mar 1997, GRANTED, Pat. No. US 6210877 Continuation-in-part of Ser. No. WO 1998-US4725, filed on 9 Mar 1998, PENDING Continuation of Ser. No. US 1997-813456, filed on 10 Mar 1997, GRANTED, Pat. No. US 6210877
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY HOAG, LLP, PATENT GROUP, WORLD TRADE CENTER WEST, 155 SEAPORT BLVD, BOSTON, MA, 02110
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Page(s)
LINE COUNT: 4114
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The kits and methods of the present invention relate to the diagnosis of cardiovascular disorders. In one aspect, the invention discloses a method and a kit for determining whether a subject has a fragile plaque disorder. In one aspect, the invention discloses a method and a kit for determining whether the subject has an occlusive disorder. In one aspect, the invention discloses a method and a kit for determining whether the subject has a restenosis disorder. Other methods of the present invention relate to the selection of therapeutics for a patient with a cardiovascular disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 27 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:237767 USPATFULL
TITLE: Genes expressed in foam cell differentiation
INVENTOR(S): Shiffman, Dov, Palo Alto, CA, UNITED STATES
Somogyi, Roland, Sydenham Ontario, CANADA
Lawn, Richard, San Francisco, CA, UNITED STATES
Seilhamer, Jeffrey J., Los Altos Hills, CA, UNITED STATES
Porter, J. Gordon, Newark, CA, UNITED STATES
Mikita, Thomas, San Francisco, CA, UNITED STATES
Tai, Julie, Cupertino, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003165924	A1	20030904
APPLICATION INFO.:	US 2002-240965	A1	20021004 (10)
	WO 2001-US11128		20010404

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-60195106	20000405

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Incyte Genopmics Inc, Legal Department, 3160 Porter Drive, Palo Alto, CA, 94304
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 3240
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to purified polynucleotides and compositions comprising pluralities of polynucleotides that are differentially expressed during foam cell development and are associated

with atherosclerosis. The present invention presents the use of the compositions as elements on a substrate, and provides methods for using the compositions and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 28 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:220740 USPATFULL
TITLE: Methods and compositions for diagnosing and treating rheumatoid arthritis
INVENTOR(S): Pittman, Debra D., Windham, NH, UNITED STATES
Feldman, Jeffrey L., Arlington, MA, UNITED STATES
Shields, Kathleen M., Harvard, MA, UNITED STATES
Trepicchio, William L., Andover, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003154032	A1	20030814
APPLICATION INFO.:	US 2001-23451	A1	20011217 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-255861P	20001215 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Patent Group, FOLEY, HOAG & ELIOT LLP, One Post Office Square, Boxton, MA, 02109
NUMBER OF CLAIMS: 40
EXEMPLARY CLAIM: 1
LINE COUNT: 25385
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides methods and compositions for diagnostic assays for detecting R.A. and therapeutic methods and compositions for treating R.A. The invention also provides methods for designing, identifying, and optimizing therapeutics for R.A. Diagnostic compositions of the invention include compositions comprising detection agents for detecting one or more genes that have been shown to be up- or down-regulated in cells of R.A. relative to normal counterpart cells. Exemplary detection agents include nucleic acid probes, which can be in solution or attached to a solid surface, e.g., in the form of a microarray. The invention also provides computer-readable media comprising values of levels of expression of one or more genes that are up- or down-regulated in R.A.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 29 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:219657 USPATFULL
TITLE: Methods for detecting and treating the early onset of aging-related conditions
INVENTOR(S): Crossman, David C., South Yorkshire, UNITED KINGDOM
Duff, Gordon W., South Yorkshire, UNITED KINGDOM
Francis, Sheila E., South Yorkshire, UNITED KINGDOM
Stephenson, Katherine, Helotes, TX, UNITED STATES
Kornman, Kenneth S., Newton, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003152947	A1	20030814
APPLICATION INFO.:	US 2002-172919	A1	20020617 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2001-298493P	20010615 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: FOLEY HOAG, LLP, PATENT GROUP, WORLD TRADE CENTER WEST, 155 SEAPORT BLVD, BOSTON, MA, 02110
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)

LINE COUNT: 3533
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Certain aspects of the invention relate to methods for determining a subject's susceptibility to the early onset or progression of aging-related conditions. In certain aspects the invention relates to accessing the genotype of a subject with respect to an allele of IL-1 pattern 1, pattern 2 and/or pattern 3. In other aspects, the invention relates to methods for selecting a therapeutic regimen, identifying age-related biomarkers, monitoring the progress of age-related conditions and identifying therapeutics for delaying or diminishing the onset of aging-related conditions.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 30 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:126723 USPATFULL
TITLE: Basal cell markers in breast cancer and uses thereof
INVENTOR(S): Botstein, David, Belmont, CA, UNITED STATES
Brown, Patrick O., Stanford, CA, UNITED STATES
Perou, Charles M., Carrboro, NC, UNITED STATES
Ring, Brian, Foster City, CA, UNITED STATES
Ross, Douglas, Burlingame, CA, UNITED STATES
Seitz, Rob, Hampton Cove, AL, UNITED STATES
van de Rijn, Jan Matthijs, LaHanda, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003086934	A1	20030508
APPLICATION INFO.:	US 2001-916849	A1	20010726 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-220967P	20000726 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Monica R. Gerber, M.D., Ph.D., Choate, Hall & Stewart, Exchange Place, 53 State Street, Boston, MA, 02109
NUMBER OF CLAIMS: 122
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Page(s)
LINE COUNT: 6518
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a variety of reagents for use in the diagnosis and management of breast cancer. The invention utilizes cDNA microarray technology to identify genes whose expression profile across a large group of tumor samples correlates with that of cytokeratin 5 and cytokeratin 17, markers for basal cells of the normal mammary lactation gland. The invention demonstrates that tumors that express cytokeratin 5/6 and/or 17 have a poor prognosis relative to tumors overall. The invention provides basal marker genes and their expression products and uses of these genes for diagnosis of breast cancer and for identification of therapies for breast cancer. In particular, the invention provides basal marker genes including cadherin3, matrix metalloproteinase 14, and cadherin EGF LAG seven-pass G-type receptor 2. The invention provides antibodies to the polypeptides expressed by these genes and methods of use thereof.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 31 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:106975 USPATFULL
TITLE: Screening methods used to identify compounds that modulate a response of a cell to ultraviolet radiation exposure
INVENTOR(S): Blumenberg, Miroslav, New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003073888	A1	20030417
APPLICATION INFO.:	US 2001-948020	A1	20010906 (9)

NUMBER DATE

PRIORITY INFORMATION: US 2000-231061P 20000908 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Charles Ashbrook, Parke Davis, Patent Department, 2800 Plymouth Road, Ann Arbor, MI, 48105
NUMBER OF CLAIMS: 108
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 13078
AB The cellular response to ultraviolet radiation exposure has been characterized on the molecular level through the use of high density gene array technology. Nucleic acid molecules and protein molecules, the expression of which are repressed or induced in response to ultraviolet radiation exposure, are identified according to a temporal pattern of altered expression post ultraviolet radiation exposure. Methods are disclosed that utilized these ultraviolet radiation-regulated molecules as markers for ultraviolet radiation exposure. Other screening methods of the invention are designed for the identification of compounds that modulate the response of a cell to ultraviolet radiation exposure. The invention also provides compositions useful for drug screening or pharmaceutical purposes.

L9 ANSWER 32 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2003:53659 USPATFULL
TITLE: Diagnostics for cardiovascular disorders
INVENTOR(S): Francis, Sheila E., Sheffield, UNITED KINGDOM
Crossman, David C., Sheffield, UNITED KINGDOM
Duff, Gordon W., Sheffield, UNITED KINGDOM
Kornman, Kenneth S., San Antonio, TX, United States
Stephenson, Katherine, San Antonio, TX, United States
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., United States (U.S. corporation)

NUMBER KIND DATE

PATENT INFORMATION: US 6524795 B1 20030225
APPLICATION INFO.: US 1999-431352 19991101 (9)
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-320395, filed on 26 May 1999, now abandoned Continuation-in-part of Ser. No. US 1997-813456, filed on 10 Mar 1997, now patented, Pat. No. US 6210877
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Myers, Carla J.
LEGAL REPRESENTATIVE: Foley Hoag LLP, Arnold, Beth, Olesen, James T.
NUMBER OF CLAIMS: 18
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 14 Drawing Figure(s); 14 Drawing Page(s)
LINE COUNT: 4006
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The kits and methods of the present invention relate to the diagnosis of cardiovascular disorders. In one aspect, the invention discloses a method and a kit for determining whether a subject has a fragile plaque disorder. In one aspect, the invention discloses a method and a kit for determining whether the subject has an occlusive disorder. In one aspect, the invention discloses a method and a kit for determining whether the subject has a restenosis disorder. Other methods of the present invention relate to the selection of therapeutics for a patient with a cardiovascular disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 33 OF 45 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2004-0141814 PASCAL
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reserved.
TITLE (IN ENGLISH): Inhibition of COX-2 in colon cancer cell lines by celecoxib increases the nuclear localization of active p53
AUTHOR: SWAMY Malisetty V.; HERZOG Christopher R.; RAO Chinthalapally V.
CORPORATE SOURCE: Chemoprevention Program, Institute For Cancer Prevention, American Health Foundation Cancer Center, Valhalla, New York 10595, United States
SOURCE: Cancer research : (Baltimore), (2003), 63(17), 5239-5242, 35 refs.
ISSN: 0008-5472 CODEN: CNREA8
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-5088, 354000113025860140
AN 2004-0141814 PASCAL
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AB Inactivation of the p53 tumor suppressor ***gene*** usually involves somatic mutation or binding of viral oncoproteins to the p53 protein. However, several types of malignant and premalignant tissues harbor a genetically wild-type, but transcriptionally inactive, form of p53, often localized in the cytoplasm. Electrophilic ***prostaglandins*** (PGs) are known to sequester and inactivate p53 in the cytoplasm, an effect that is likely to occur when ***cyclooxygenase*** (COX)-2 levels become elevated during colon carcinogenesis. We determined the localization and ***expression*** of p53 in the presence of PGA.sub.1 and celecoxib, a selective COX-2 inhibitor in ***human*** colon cell lines HCT-116 (wild-type p53) and HT-29 (***mutant*** p53). In the absence of treatment, p53 protein accumulated preferentially in the nucleus in both cell lines. We observed that the total cellular levels of p53 protein increased with exposure time and concentration of PGA.sub.1. By contrast, p21 protein levels remained unchanged as a function of time and concentration of PGA.sub.1. In the presence of 20 .mu.M PGA.sub.1, p53 accumulated preferentially in the cytosol. The nuclear:cytosol ratios of p53 were 31 and 2.1 in the controls and in the presence of PGA.sub.1 in HCT-116 cells but were 22 and 4, respectively, in HT-29 cells. Treatment with 50 .mu.M celecoxib for 24 h did not significantly change p53 ***expression*** and localization. However, in the presence of 100 .mu.M celecoxib, p53 levels increased in the nucleus. The nuclear:cytosol ratios were then 31 (control) and 60 (100 .mu.M celecoxib) in HCT-116 cells and 22 (control) and 36 (100 .mu.M celecoxib) in HT-29 cells. These results indicate that electrophilic PGs cause wild-type p53 accumulation in the cytosol where it is inactive. Inhibition of COX-2 by celecoxib appears to alleviate this effect on p53 by reducing electrophilic PG synthesis. Thus, COX-2 inhibition of electrophilic PG formation appears to protect p53 tumor suppressor function.

L9 ANSWER 34 OF 45 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2003-0157085 PASCAL
COPYRIGHT NOTICE: Copyright .COPYRGT. 2003 INIST-CNRS. All rights reserved.
TITLE (IN ENGLISH): Tissue differential microarray analysis of dexamethasone induction reveals potential mechanisms of steroid glaucoma
AUTHOR: LO Wayne R.; ROWLETTE Laura Leigh; CABALLERO Montserrat; PING YANG; HERNANDEZ M. Rosario; BORRAS Teresa
CORPORATE SOURCE: Department of Ophthalmology, Duke University Medical Center, Durham, North Carolina, United States; Department of Ophthalmology and Visual Sciences, Washington University School of Medicine, St. Louis, Missouri, United States; Department of Anatomy and Neurobiology, Washington University School of Medicine, St. Louis, Missouri, United States; Department of Genetics, Duke University Medical Center, Durham, North Carolina, United States

SOURCE: Investigative ophthalmology & visual science, (2003), 44(2), 473-485, 69 refs.
ISSN: 0146-0404 CODEN: IOVSDA
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-12095, 354000103785040040
AN 2003-0157085 PASCAL

AB PURPOSE. To identify myocilin (TIGR/MYOC) properties that are specific to the ***human*** trabecular meshwork (HTM). To search for genes highly ***expressed*** in dexamethasone (DEX> induced HTM cells that are barely ***expressed*** or absent in DEX-induced cells from other tissues. METHODS. TIGR/MYOC induction by DEX (10.sup.-.sup.7 M for 8-10 days) was analyzed by Northern and Western blot analyses in HTM, ***human*** umbilical vein endothelial cells, HeLa cells, and ***human*** embryonic skeletal muscle cells and optic nerve head (ONH) astrocytes at confluence. Processing and secretion were analyzed after the cells were infected with adenoviruses over- ***expressing*** wild-type and ***mutant*** forms of TIGR/MYOC. Affymetrix U95Av2 GeneChips (n = 6) and software were used to compare paired ***expression*** profiles of HTM, HTM-DEX, ONH astrocytes, and ONH astrocytes-DEX. Identification of HTM-DEX-specific genes (compared with ONH astrocytes-DEX) was performed by selecting genes with the highest fold change values (>=20). Genes with fold change values of four or more were matched with loci linked to glaucoma, by using ***gene*** databases. RESULTS. TIGR/MYOC induction by DEX occurred only in HTM cells. Secretory and glycosylation characteristics remained the same across cell types. ***Expression*** profile analysis revealed multiple genes differentially upregulated in HTM-DEX including, in addition to TIGR/MYOC, a serine protease inhibitor (.alpha.1-antichymotrypsin), a neuroprotective factor (pigment epithelium-derived factor), an antiangiogenesis factor (cornea-derived transcript 6), and a ***prostaglandin*** ***synthase*** (***prostaglandin*** D.sub.2 synthase). Fifteen of the 249 genes with fold change values of four or more mapped to glaucoma-linked loci. CONCLUSIONS. The induction of TIGR/MYOC by DEX is HTM-specific, whereas its secretory and glycosylation characteristics are ubiquitous. The known functions of HTM-DEX-specific genes reveal the presence of protective and damaging mechanisms for regulation of IOP during DEX treatment. Besides TIGR/MYOC, other HTM-DEX-specific genes may be good candidates for linkage to glaucoma.

L9 ANSWER 35 OF 45 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:354204 CAPLUS
DOCUMENT NUMBER: 136:336218
TITLE: Animal cell lines expressing the gene for a splicing isoenzyme of prostaglandin synthase for use in drug screening and other therapeutic applications
INVENTOR(S): Anger Leroy, Marielle; Hanf, Remi
PATENT ASSIGNEE(S): Laboratoire Innothera, Fr.
SOURCE: Fr. Demande, 63 pp.
CODEN: FRXXBL
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
FR 2811677	A1	20020118	FR 2000-9139	20000712
FR 2811677	B1	20041224		

PRIORITY APPLN. INFO.: FR 2000-9139 20000712
AB Animal cell lines expressing the gene for an isoenzyme of prostaglandin synthase that arose through differential splicing affecting exons 3 and 9 are described. These lines produce prostaglandin D2 and 15-deoxy-.DELTA.12-14 prostaglandin J2 from arachidonic acid and so may be used to screen for inhibitors. The enzyme is inhibited by non-steroidal anti-inflammatory drugs and so may be used to screen for antiinflammatories. A cDNA for the isoenzyme was cloned by PCR using

primers derived from the known sequence of the PGHS1 gene. Most of the clones obtained corresponded to the known sequence of the human prostaglandin synthase. One of the clones showed a 111bp deletion affecting exons 3 and 9. The gene was stably expressed in CHO-K1 cells using the prior art expression vector pDR2EF1.alpha..

L9 ANSWER 36 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2002:171875 USPATFULL
TITLE: Gene markers useful for detecting skin damage in response to ultraviolet radiation
INVENTOR(S): Blumenberg, Miroslav, New York, NY, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002090624	A1	20020711
	US 6794137	B2	20040921
APPLICATION INFO.:	US 2001-947870	A1	20010906 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-231454P	20000908 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HALE AND DORR, LLP, 60 STATE STREET, BOSTON, MA, 02109
NUMBER OF CLAIMS: 97
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 10110
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The cellular response to ultraviolet radiation exposure has been characterized on the molecular level through the use of high density gene array technology. Nucleic acid molecules and protein molecules, the expression of which are repressed or induced in response to ultraviolet radiation exposure, are identified according to a temporal pattern of altered expression post ultraviolet radiation exposure. Methods are disclosed that utilized these ultraviolet radiation-regulated molecules as markers for ultraviolet radiation exposure. Other screening methods of the invention are designed for the identification of compounds that modulate the response of a cell to ultraviolet radiation exposure. The invention also provides compositions useful for drug screening or pharmaceutical purposes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 37 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2002:27104 USPATFULL
TITLE: ATHEROSCLEROSIS-ASSOCIATED GENES
INVENTOR(S): JONES, KAREN ANNE, ESSEX, UNITED KINGDOM
VOLKMUTH, WAYNE, MENLO PARK, CA, UNITED STATES
WALKER, MICHAEL G., SUNNYVALE, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002015950	A1	20020207
APPLICATION INFO.:	US 1999-349015	A1	19990707 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LEGAL DEPARTMENT, INCYTE GENOMICS, INC., 3160 PORTER DRIVE, PALO ALTO, CA, 94304
NUMBER OF CLAIMS: 20
EXEMPLARY CLAIM: 1
LINE COUNT: 3426
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides novel atherosclerosis-associated polynucleotides and polypeptides encoded by those genes. The invention also provides expression vectors, host cells, and antibodies. The invention also provides methods for screening or purifying ligands and diagnosing, treating or preventing atherosclerosis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 38 OF 45 USPATFULL on STN
ACCESSION NUMBER: 2002:16917 USPATFULL
TITLE: Growth stimulation of biological cells and tissue by electromagnetic fields and uses thereof
INVENTOR(S): Wolf, David A., Houston, TX, UNITED STATES
Goodwin, Thomas J., Friendswood, TX, UNITED STATES
PATENT ASSIGNEE(S): National Aeronautics & Space Administration (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002009797	A1	20020124
	US 6673597	B2	20040106

APPLICATION INFO.: US 2001-798854 A1 20010228 (9)
RELATED APPLN. INFO.: Division of Ser. No. US 2000-587028, filed on 2 Jun 2000, ABANDONED
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: NASA JOHNSON SPACE CENTER, MAIL CODE HA, 2101 NASA RD 1, HOUSTON, TX, 77058
NUMBER OF CLAIMS: 37
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 11 Drawing Page(s)
LINE COUNT: 1200
AB The present invention provides systems for growing two or three dimensional mammalian cells within a culture medium facilitated by an electromagnetic field, and preferably, a time varying electromagnetic field. The cells and culture medium are contained within a fixed or rotating culture vessel, and the electromagnetic field is emitted from at least one electrode. In one embodiment, the electrode is spaced from the vessel. The invention further provides methods to promote neural tissue regeneration by means of culturing the neural cells in the claimed system. In one embodiment, neuronal cells are grown within longitudinally extending tissue strands extending axially along and within electrodes comprising electrically conductive channels or guides through which a time varying electrical current is conducted, the conductive channels being positioned within a culture medium.

L9 ANSWER 39 OF 45 PASCAL COPYRIGHT 2006 INIST-CNRS. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2002-0389952 PASCAL

TITLE (IN ENGLISH): COX selectivity and animal models for colon cancer
COX II inhibitors
AUTHOR: OSHIMA Masanobu; TAKETO Makoto M.
BROOKS Peter (ed.)
CORPORATE SOURCE: Department of Pharmacology, Kyoto University Graduate School of Medicine, Yoshida-Konoe cho, Sakyo-ku, Kyoto 606-8501, Japan
University of Queensland, Edith Cavell Building, Royal Brisbane Hospital, Herston Qld 4029, Australia
SOURCE: Current pharmaceutical design, (2002), 8(12), 1021-1034, 160 refs.
ISSN: 1381-6128
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: Netherlands
LANGUAGE: English
AVAILABILITY: INIST-26320, 354000101026560010
AN 2002-0389952 PASCAL

AB Early experiments performed during 1980s and 1990s using carcinogen-induced rat intestinal tumor models demonstrated the inhibitory effects of non-steroidal anti-inflammatory drugs (NSAIDs) on intestinal tumorigenesis. Furthermore, epidemiological studies and clinical trials for familial adenomatous polyposis (FAP) patients supported the possibility that NSAIDs can be used as chemopreventive

agents. The major target molecules of NSAIDs are cyclooxygenases (COX), which catalyze the rate-limiting step of ***prostaglandin*** biosynthesis. Two isoenzymes of COX have been identified; ***COX*** - ***1*** and COX-2. Whereas ***COX*** - ***1*** is ***expressed*** constitutively in most tissues and responsible for tissue homeostasis, COX-2 is inducible and plays an important role in inflammation and intestinal tumorigenesis. A genetic study using compound ***mutant*** mice of COX-2.sup.-.sup./.sup.-, and Apc.sup..DELTA..sup.7.sup.1.sup.6 which is a model for ***human*** familial adenomatous polyposis (FAP), directly demonstrated that induction of COX-2 is critical for intestinal polyp formation. Numerous studies have also demonstrated that COX-2 selective inhibitors suppress intestinal polyp formation in Apc ***gene*** - ***mutant*** mice, and xenografted cancer cell growths. In addition, stimulation of angiogenesis is one of the major effects by COX-2 ***expression*** that is induced in the polyp stromal cells. On the other hand, another study indicated that ***COX*** - ***1*** also plays an important role in the early stage of intestinal tumorigenesis. These data from animal model studies should be helpful in understanding the in vivo mechanism(s) of tumor suppression by NSAIDs or COX-2 inhibitors. Here, we review the animal studies that have been published as of August 2001, and reported to suppress intestinal tumor growths by NSAIDs or COX-2 inhibitors.

L9 ANSWER 40 OF 45 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2001250669 ESBIOBASE

TITLE: Phorbol 12-myristate 13-acetate triggers the protein kinase A-mediated phosphorylation and activation of the PDE4D5 cAMP phosphodiesterase in human aortic smooth muscle cells through a route involving extracellular signal regulated kinase (ERK)

AUTHOR: Baillie G.; Mackenzie S.J.; Houslay M.D.

CORPORATE SOURCE: Dr. M.D. Houslay, Division of Biochemistry, Davidson and Wolfson Buildings, University of Glasgow, Glasgow G12 8QQ, United Kingdom.
E-mail: m.houslay@bio.gla.ac.uk

SOURCE: Molecular Pharmacology, (2001), 60/5 (1100-1111), 48 reference(s)
CODEN: MOPMA3 ISSN: 0026-895X

DOCUMENT TYPE: Journal; Article

COUNTRY: United States

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Phosphodiesterase 4D5 is the sole PDE4D cAMP phosphodiesterase isoform ***expressed*** in ***human*** aortic smooth muscle cells (HASMC). Phorbol 12-myristate 13-acetate (PMA) challenge of HASMC rapidly activated PDE4D5 through a process ablated by the mitogen-activated protein kinase kinase inhibitor PD98059. PMA elicited an inhibitory effect on PDE4D5 activity in HASMC treated with the ***cyclooxygenase*** (COX) inhibitor indomethacin, the COX-2 selective inhibitor NS-398, the phospholipase A.sub.2 inhibitor quinacrine, and the cAMP-dependent protein kinase A (PKA) inhibitor H89. PMA challenge of COS-1 cells elicited the rapid inhibition and phosphorylation of both ***recombinant*** and endogenous PDE4D5 in a manner ablated by PD98059 and not seen in S651A ***mutant*** PDE4D5. PMA promoted the generation of PGE.sub.2 in the medium of HASMC and caused activation of both extracellular signal-regulated kinase (ERK) and PKA through a process ablated by indomethacin, NS-398, quinacrine, and PD98059. Exogenous ***prostaglandin*** (PG) E.sub.2 increased cAMP levels and activated PKA in HASMC. COX-2 was ***expressed*** in HASMC but not in COS-1 cells. Forskolin challenge of COS-1 cells activated PDE4D5 by causing the PKA-mediated phosphorylation of Ser126 as detected using a novel phosphospecific antiserum. PMA challenge of HASMC elicited phosphorylation of the stimulatory PKA-specific phosphorylation site, Ser126 in PDE4D5 in a manner ablated by PD98059, indomethacin, and H89. We propose that, in HASMC, PMA activates PDE4D5 through an ERK-controlled autocrine mechanism. This involves PGE.sub.2 generation, which causes activation of adenylyl cyclase, allowing PKA to elicit net activation of PDE4D5 by phosphorylation at Ser126.

L9 ANSWER 41 OF 45 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 2000254351 ESBIOBASE
TITLE: Suppression of intestinal polyposis in Apc(.DELTA.716) knockout mice by an additional mutation in the cytosolic phospholipase A.sub.2 gene
AUTHOR: Takaku K.; Sonoshita M.; Sasaki N.; Uozumis N.; Doi Y.; Shimizu T.; Taketo M.M.
CORPORATE SOURCE: M.M. Taketo, Laboratory of Biomedical Genetics, Graduate Sch. of Pharmaceut. Sci., University of Tokyo, 7-3-1 Hongo, Bunkyo-Ku, Tokyo 113-0033, Japan. E-mail: taketo@mol.f.u-tokyo.ac.jp
SOURCE: Journal of Biological Chemistry, (03 NOV 2000), 275/44 (34013-34016), 41 reference(s)
CODEN: JBCHA3 ISSN: 0021-9258
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Arachidonic acid is a precursor for biosynthesis of eicosanoids, including ***prostaglandins*** , thromboxanes, leukotrienes, and lipoxins. Cytosolic phospholipase A.sub.2 (cPLA.sub.2) plays a key role in the release of arachidonic acid as the substrate of ***cyclooxygenase*** - ***1*** (***COX*** - ***1***) or COX-2. We found that the level of cPLA.sub.2 mRNA was markedly elevated in the polyps and correlated with the polyp size in the small intestine of the Apc(.DELTA.716) knockout mouse, a model for ***human*** familial adenomatous polyposis. To determine the role of cPLA.sub.2 in intestinal tumorigenesis, we then introduced a cPLA.sub.2 ***gene*** mutation into Apc(.DELTA.716) mice. In the compound ***mutant*** mice, the size of the small intestinal polyps was reduced significantly, although the numbers remained unchanged. These results provide direct genetic evidence that cPLA.sub.2 plays a key role in the expansion of polyps in the small intestine rather than in the initiation process. In contrast, colonic polyps were not affected in either size or number. Interestingly, group X sPLA.sub.2 was constitutively ***expressed*** in the colon at much higher levels than in the small intestine. These results suggest that in the colon, group X sPLA.sub.2 supplies arachidonic acid in both the normal epithelium and the polyps even in the absence of cPLA.sub.2.

L9 ANSWER 42 OF 45 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1999047663 ESBIOBASE
TITLE: Antagonistic effects of an alternative splice variant of human IL-4, IL- 4.delta.2, on IL-4 activities in human monocytes and B cells
AUTHOR: Arinobu Y.; Atamas S.P.; Otsuka T.; Niiro H.; Yamaoka K.; Mitsuyasu H.; Niho Y.; Hamasaki N.; White B.; Izuhara K.
CORPORATE SOURCE: Y. Arinobu, Department of Clinical Chemistry, Laboratory Medicine, Kyushu University, Fukuoka 812-8582, Japan.
SOURCE: Cellular Immunology, (01 FEB 1999), 191/2 (161-167), 44 reference(s)
CODEN: CLIMB8 ISSN: 0008-8749
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB IL-4 is a pleiotropic cytokine which exerts its actions on various lineages of hematopoietic and nonhematopoietic cells. This cytokine is one of the central regulators of immunity in health and disease states. An alternative ***splice*** ***variant*** , in which the second of four exons is omitted, has been recently described and designated as IL-4.delta.2. The ***variant*** has been previously described as a potential naturally occurring antagonist of ***human*** IL-4 (hIL-4)-stimulated T cell proliferation. In this study, we investigated the effects of ***recombinant*** ***human*** (rh) IL-4.delta.2 on monocytes and B cells. In monocytes, rhIL-4.delta.2 blocked inhibitory

action of hIL-4 on LPS-induced ***cyclooxygenase*** -2 ***expression*** and subsequent ***prostaglandin*** E.sub.2 secretion. In B cells, rhIL-4.delta.2 was an antagonist of the hIL-4-induced synthesis of IgE and ***expression*** of CD23. Our results broaden the spectrum of hIL-4-antagonistic activities of rhIL-4.delta.2, thus creating the background for the potential use of rhIL-4.delta.2 as a therapeutic anti-hIL-4 agent.

L9 ANSWER 43 OF 45 USPATFULL on STN
ACCESSION NUMBER: 1998:131579 USPATFULL
TITLE: Methods and composition for in vivo gene therapy
INVENTOR(S): Debs, Robert James, Mill Valley, CA, United States
Zhu, Ning, El Cerrito, CA, United States
PATENT ASSIGNEE(S): The Regents of the University of California, Oakland, CA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5827703		19981027
APPLICATION INFO.:	US 1994-246376		19940519 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1992-992687, filed on 17 Dec 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-927200, filed on 6 Aug 1992, now abandoned which is a continuation-in-part of Ser. No. US 1992-894498, filed on 4 Jun 1992, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Rories, Charles C.P.
LEGAL REPRESENTATIVE: Townsend and Townsend and Crew LLP
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 84 Drawing Figure(s); 68 Drawing Page(s)
LINE COUNT: 2448
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel methods and compositions are provided for introducing a gene capable of modulating the genotype and phenotype into two or more tissues following systemic administration. The gene can be introduced into a mammalian host by way of an expression vector either as naked DNA or complexed to lipid carriers, particularly cationic lipid carriers. Multiple individual tissues can be transfected using naked DNA. Using a DNA: lipid carrier complex, multiple tissues and cell types can be transfected. The techniques and compositions find use in the palliation or treatment of any of a variety of genetic-based disorders.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L9 ANSWER 44 OF 45 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1995033490 ESBIOBASE
TITLE: Prostaglandin H synthase-1: Evaluation of C-terminus function
AUTHOR: Ren Y.; Loose-Mitchell D.S.; Kulmacz R.J.
CORPORATE SOURCE: R.J. Kulmacz, Division of Hematology, Department of Internal Medicine, Univ. of Texas Health Science Center, 6431 Fannin St., Houston, TX 77030, United States.
SOURCE: Archives of Biochemistry and Biophysics, (1995), 316/2 (751-757)
CODEN: ABBIA4 ISSN: 0003-9861
DOCUMENT TYPE: Journal; Article
COUNTRY: United States
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The first committed step in ***prostaglandin*** biosynthesis is catalyzed by ***prostaglandin*** H synthase, an enzyme localized to the endoplasmic reticulum (ER) membrane in a variety of cells. Several types of C-terminal region peptide ***sequence*** motifs have been found to lead to ER retention of other proteins. We have tested the potential role of such signals in the ER localization and catalytic activity of ***human*** isoform-1 of the synthase (PGHS-1). PGHS-1

mutants with alterations in the C-terminus designed to disrupt potential retention signals were ***expressed*** in transfected COS-1 cells. The mutations included: substitution of valine for the ultimate leucine residue (position 600) to disrupt a KDEL-type signal, substitution of a neutral glutamine for arginine at position 595 to disrupt signals based on positive charge, and deletion of the last six residues, to remove all of the wild-type extreme C-terminus. The subcellular localization of each ***recombinant*** PGHS-1 was assessed by differential centrifugation and by immunofluorescence microscopy. None of the mutations led to a significant change in the distribution of PGHS-1 between microsomes and other cellular fractions. Immunostaining of wild-type PGHS-1 and all of the ***mutants*** colocalized with that of protein disulfide isomerase, an ER marker protein. However, mutation of the terminal leucine or deletion of the last six residues did lead to loss of ***cyclooxygenase*** activity. Mutation of the terminal leucine also altered the pattern of fragments produced by limited proteolysis, indicating that this mutation led to changes in the polypeptide folding which might account for the loss of activity. The results indicate that the extreme C-terminal region is important to the functional integrity of PGHS-1, but it is not an essential part of the intracellular targeting mechanism.

L9 ANSWER 45 OF 45 BIOTECHNO COPYRIGHT 2006 Elsevier Science B.V. on STN
ACCESSION NUMBER: 1990:20237823 BIOTECHNO
TITLE: Role of the Ha-ras (Ras(H)) oncogene in mediating progession of the tumor cell phenotype (Review)
AUTHOR: Boylan J.F.; Jackson J.; Steiner M.R.; Shih T.Y.; Duigou G.J.; Roszman T.; Fisher P.B.; Zimmer S.G.
CORPORATE SOURCE: Microbiology/Immunology Dept., University of Kentucky, Medical Center,Lexington, KY 40536-0084, United States.
SOURCE: Anticancer Research, (1990), 10/3 (717-724)
CODEN: ANTRD4 ISSN: 0250-7005
DOCUMENT TYPE: Journal; General Review
COUNTRY: Greece
LANGUAGE: English
SUMMARY LANGUAGE: English
AN 1990:20237823 BIOTECHNO
AB Recent experimental evidence indicates that the c-Ha-ras (ras(H)) oncogene may be causually involved in the etiology and evolution of specific ***human*** neoplasms. In addition, cultured cells transformed by the ras(H) oncogene can induce both a tumorigenic and metastatic phenotype when ***expressed*** in appropriate cultured cells. To begin to define the molecular and biochemical mechanism(s) by which the ras(H) oncogene induce their effects on ***expression*** of the transformed state we have employed a cloned rat embryo fibroblast (CREF) cell line. Transformation of CREF cells with wild-type 5 adenovirus (Wt) results in transformed cells which display anchorage-independence and an increased saturation density in monolayer culture, but are non-tumorigenic in both athymic nude mice and syngeneic Fischer rats. In contrast, when CREF cells are transformed with ***mutant*** type 5 adenoviruses, such as H5hrl, or the E1A transforming ***gene*** from hrl (0-4.5), tumors are induced in both nude mice and syngeneic rats. However, hrl (0-4.5)-transformed CREF cells are not metastatic following intravenous injection into the tail vein of syngeneic rats. Insertion of an activated T24 ras(H) oncogene or a wild-type v-ras(H) oncogene into CREF, wt-transformed CREF or hrl (0-4.5)-transformed CREF cells results in acquisition of a metastatic phenotype by these cells. A ***mutant*** v-ras(H) oncogene (***mutant*** 116K), which is defective in GTP binding and the induction of transformation of NIH 3T3 cells, does not induce transformation in CREF cells, but it can progress wt-transformed CREF cells to a tumorigenic-non-metastatic state. Employing this model system which displays well-defined and stable stages in the tumor cell progression lineage, we have analyzed the potential role of changes in the phosphatidylinositol (PI) cycle and phospholipase A.sub.2 (PLA.sub.2) enzyme activity during progression to a tumorigenic and metastatic phenotype. An increase in PI cycle intermediates (primarily inositol triphosphate; IP.sub.3) were observed only in the wt-transformed and hrl (0-4.5)-transformed CREF cell lines transfected with the ras(H) oncogene.

In the case of PLA.sub.2, all ras(H)-transformed CREF cell lines displayed increased activity. In contrast, CREF cells transformed only by Ad5 (Wt or hrl (0-4.5)) or the 116K v-ras(H) oncogene did not display increased PLA.sub.2 activity similar to that observed in ras(H) transfected cells. Since one important metabolite generated by PLA.sub.2 is arachidonic acid, which is converted into ***prostaglandins*** and leukotrienes by ***cyclooxygenase*** or lipooxygenase, respectively, the levels of ***prostaglandin*** E.sub.2 (PGF.sub.2) in the various cell lines were monitored. The pattern of secreted PGE.sub.2 levels for both the tumorigenic and metastatic cell lines was identical to that observed for PLA.sub.2 activity. A selective increase of PGE.sub.2 as opposed to IP.sub.3 levels as a result of ***expression*** of the ras(H) oncogene was observed in CREF cells transformed with a ras(H) oncogene controlled by a metallothionein promoter. The present studies indicate that the ras(H) oncogene-mediated alterations required to induce changes in progression of the transformed phenotype in CREF cells depend on the genetic background of the target cell. In addition, PLA.sub.2 and its resultant metabolites may play an important role in the process of progression of the transformed state.

=> d his

L1 QUE ((CYCLOOXYGENASE(W) 1) OR CYCLOOXYGENASE OR COX-1 OR (COX(W

L2 236717 S L1
L3 12786 S (GENE OR SEQUENCE OR POLYNUCLEOTIDE OR CLONE OR RECOMBINANT)(
L4 5814 DUP REM L3 (6972 DUPLICATES REMOVED)
L5 3424 S (VECTOR# OR EXPRESS? OR HOST)(S)L4
L6 863 S PROSTAGLANDIN# (S)L5
L7 72 S (MUTANT# OR VARIANT# OR SPLIC?)(S)L6
L8 45 S (HUMAN OR (HOMO(W)SAPIENS))(S)L7
L9 45 DUP REM L8 (0 DUPLICATES REMOVED)

=> log y